Refine Search

Search Results -

Terms	Documents
L13 same L10	10

Database:
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Derwent World Patents Index
IBM Technical Disclosure Bulletins

Search: L16

Refine Search

Recall Text | Clear | Interrupt

Search History

DATE: Thursday, July 08, 2004 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI; PLUR=YES; OP=ADJ			
L16	L13 same l10	10	L16
L15	l13 same l6	0	L15
L14	L13 same l2	0	L14
L13	plant with carcinogen	108	L13
L12	l10 and l1	0	L12
L11	L10 same l1	0	L11
L10	gene	270954	L10
L9	l6 and l1	0	L9
L8	L6 same l1	0	L8
L7	L6 same l1	0	L7
L6	mutation or mutant	101601	L6
L5	l2 and l1	0	L5
L4	l2 same l1	0	L4
L3	L2 with l1	0	L3

L2	transgenic	43476	L2
L1	plant with anthracene	53	L1

END OF SEARCH HISTORY

First Hit Previous Doc Next Doc Go to Doc#

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L16: Entry 2 of 10 File: PGPB Jun 19, 2003

DOCUMENT-IDENTIFIER: US 20030113710 A1
TITLE: Methods for identifying genes essential to the growth of an organism

Summary of Invention Paragraph:
[0002] Identification, sequencing and characterization of genes is a major goal of modem scientific research. By identifying genes, determining their sequences and characterizing their biological function, it is possible to employ recombinant technology to produce large quantities of valuable gene products, e.g. proteins and peptides. Additionally, knowledge of gene sequences can provide a key to diagnosis, prognosis and treatment in a variety of infectious diseases and disease states in plants and animals which are characterized by inappropriate expression and/or repression of selected genes or by the influence of external factors, e.g., carcinogens or teratogens, on gene function.

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(FILE 'HOME' ENTERED AT 11:57:51 ON 08 JUL 2004)

FILE 'MEDLINE' ENTERED AT 11:57:57 ON 08 JUL 2004

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L1            42 S S. TYPHIMURIUM  AND YEAST
L2          5916 S ANTHRACENE
L3             0 S L2 AND L1
L4         79652 S YEAST
L5            23 S L4 AND L2
L6            23 DUP REM L5 (0 DUPLICATES REMOVED)
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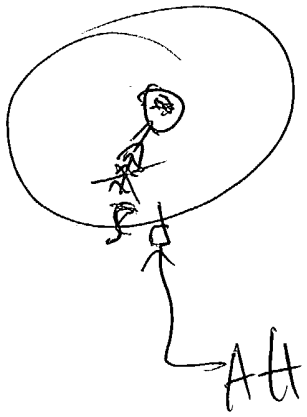
FILE 'MEDLINE, CANCERLIT, BIOTECHDS, CAPLUS, BIOSIS, EMBASE' ENTERED AT 12:03:03 ON 08 JUL 2004

```
L7         81540 S L2
L8        492119 S L4
L9       1511133 S MUTATION OR MUTAN?
L10      3497842 S GENE
L11       786993 S L10 AND L9
L12          210 S L11 AND 8 AND L7
L13           96 DUP REM L12 (114 DUPLICATES REMOVED)
L14      1541211 S PLANT
L15            1 S L14 AND L13
L16      5442700 S IN VITRO OR CULTU?
L17          242 S L16 AND  L9 AND L10 AND L7
L18          111 DUP REM L17 (131 DUPLICATES REMOVED)
L19      2007374 S YEAST OR PLANT
L20            9 S L19 AND L18
L21           46 S L18 AND (ASSA? OR IDENTIF?)
L22      1541211 S PLANT
L23         2148 S L22 AND L7
L24       786993 S L9 AND L10
L25            9 S L24 AND L23
L26            8 DUP REM L25 (1 DUPLICATE REMOVED)
L27       232413 S TRANSGENIC
L28            9 S L27 AND L23
L29            6 DUP REM L28 (3 DUPLICATES REMOVED)
L30           40 S L23 AND L9
L31           26 DUP REM L30 (14 DUPLICATES REMOVED)
L32           58 S L23 AND L10
L33           41 DUP REM L32 (17 DUPLICATES REMOVED)
```

=>

L31 ANSWER 11 OF 26 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1998:538180 CAPLUS
DN 129:270149
TI Antimutagenic constituents of Casimiroa edulis with potential cancer chemopreventive activity
AU Ito, Aiko; Shamon, Lisa A.; Yu, Boyang; Mata-Greenwood, Eugenia; Lee, Sang Kook; Van Breemen, Richard B.; Mehta, Rajendra G.; Farnsworth, Norman R.; Fong, Harry H. S.; Pezzuto, John M.; Kinghorn, A. Douglas
CS Program for Collaborative Research in the Pharmaceutical Sciences and Department of Medicinal Chemistry and Pharmacognosy College of Pharmacy, University of Illinois at Chicago, Chicago, IL, 60612, USA
SO Journal of Agricultural and Food Chemistry (1998), 46(9), 3509-3516
CODEN: JAFCAU; ISSN: 0021-8561
PB American Chemical Society
DT Journal
LA English
AB An Et acetate extract derived from the seeds of the medicinal and food **plant** C. edulis inhibited mutagenicity induced by 7,12-dimethylbenz[a]**anthracene** (DMBA) with Salmonella typhimurium strain TM677. It also showed complete inhibition of DMBA-induced preneoplastic lesions with an in vitro mouse mammary gland organ culture system at a concentration of 10 μg/mL. Bioassay-guided phytochem. investigation of this extract using antimutagenicity as a monitor led to the isolation of 4 furocoumarins, constituted by the known compds. phellopterin (1) and isopimpinellin (2) and the novel compds. (R,S)-5-methoxy-8-[(6,7-dihydroxy-3,7-dimethyl-2-octenyl)oxy]psoralen (3) and (R,S)-8-[(6,7-dihydroxy-3,7-dimethyl-2-octenyl)oxy]psoralen (4). Four known alkaloids, casimiroin (5), 4-methoxy-1-methyl-2(1H)-quinolinone (6), 5-hydroxy-1-methyl-2-phenyl-4-quinolone (7), and γ-fagarine (8), and 2 known flavonoids, zapotin (9) and 5,6,2'-trimethoxyflavone (10), were also isolated. Of these isolates, compds. 3 and 5 showed the most potent antimutagenic effects in the forward mutagen assay utilizing S. typhimurium strain TM677, whereas casimiroin (5) and 5,6,2'-trimethoxyflavone (10) significantly inhibited the formation of DMBA-induced preneoplastic lesions in mouse mammary gland organ culture.



L21 ANSWER 11 OF 46 MEDLINE on STN
AN 91000223 MEDLINE
DN PubMed ID: 2119594
TI Relationship between chemically induced Ha-ras **mutation** and transformation of BALB/c 3T3 cells: evidence for chemical-specific activation and cell type-specific recruitment of oncogene in transformation.
AU Nakazawa H; Aguelon A M; Yamasaki H
CS International Agency for Research on Cancer, Lyon, France.
NC RO1 CA40534 (NCI)
SO Molecular carcinogenesis, (1990) 3 (4) 202-9.
Journal code: 8811105. ISSN: 0899-1987.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199011
ED Entered STN: 19910117
Last Updated on STN: 19970203
Entered Medline: 19901105
AB BALB/c 3T3 cells were exposed to 7,12-dimethylbenz[a]**anthracene** (DMBA) and resultant transformed foci were analyzed for the presence of A182----T **mutation** at codon 61 of Ha-ras (a **mutation** found in many DMBA-induced animal tumors). None of the 30 independently cloned transformed cell lines contained such a **mutation**. In order to see whether DMBA is able to induce this **mutation** in BALB/c 3T3 cells, we developed a method sensitive enough to detect this specific **mutation** at the frequency of 10(-6). Employing this **assay**, we found time- and dose-dependent induction by DMBA of Ha-ras A182----T **mutation** in BALB/c 3T3 cells; for example, 2 wk after exposure to 100 micrograms/mL DMBA, 1.4 in 1 X 10(4) cells contained this specific **mutation**. On the other hand, other agents that also induce BALB/c 3T3 cell transformation, such as 3-methylcholanthrene (MCA), 12-O-tetradecanoylphorbol-13-acetate (TPA), N-methyl-N'-nitro-N-nitrosoguanidine (MNNG), or ultraviolet light, did not induce the **mutation** at detectable frequency (less than 10(-6)). These results suggest that DMBA efficiently induces Ha-ras **mutation** in BALB/c 3T3 cells but that this **mutation** is not recruited in the process of cell transformation. A hypothesis of carcinogen-specific **mutation** of Ha-ras **gene** and its tissue (cell type)-specific recruitment in carcinogenesis is proposed.

L21 ANSWER 15 OF 46 CANCERLIT on STN
AN 93690104 CANCERLIT
DN 93690104
TI Molecular analysis of forward and reverse somatic mutations at the human APRT locus.
AU Zhu Y
CS Indiana Univ.
SO Diss Abstr Int [B], (1992) 53 (6) 2643.
ISSN: 0419-4217.
DT (THESIS)
LA English
FS Institute for Cell and Developmental Biology
EM 199305
ED Entered STN: 19941107
Last Updated on STN: 19941107
AB Somatic cell **mutation** plays an important role in the etiology of a number of genetic diseases and many types of cancer (eg, retinoblastoma and Wilms' tumor). This thesis describes the use of the human adenine phosphoribosyltransferase (APRT) **gene** in cell **culture** for study of the nature and consequences of spontaneous and induced mutations at a typical mammalian locus. Two APRT-deficient human cell lines were analyzed. An ICR170H-induced fibrosarcoma cell line, HTD114, was shown to have a single G insertion in an existing string of 5Gs in exon 2 of one allele and in exon 3 of the second allele. A hepatoma cell line, 3B225H, was shown to have a G to C transversion at the 3rd bp of intron 1, which results in very low levels of normal APRT mRNA. With an APRT reversion **assay** in HTD114 cells, mutagens and carcinogens (such as mitomycin C, benzo[a]pyrene diol epoxide, 2-aminoanthracene, 7,12-dimethylbenz[a]**anthracene** and aflatoxin B1) were demonstrated to induce a single base pair deletion that resulted in frame restoration. The APRT reversion rates induced by these chemicals, except for mitomycin C (1.2-3.3 x 10(-5)), are about 10(3)-fold increased over the rate of spontaneous reversion. Rat liver homogenate coupled with NADPH cofactors was an essential activation system for some mutagenesis. However, mitomycin C and other mutagens produced little effect on the induction of interallelic mitotic recombination. By selection and analysis of Aprt- **mutants** in a human cell line heterozygous at APRT, the molecular mechanisms of loss of APRT activity were investigated. These included loss of the wild-type allele (62%) and intragenic mutations (38%). Lost of the wild-type APRT allele was frequently accompanied by loss of the relatively close proximal marker D16S77, but not the more distant proximal marker D16S4. These data indicate that high-frequency mitotic recombination or deletion occurred at the region between D16S77 and D16S4 on chromosome 16. Also, point mutations were demonstrated to be responsible for the loss of APRT activity in other clones. These demonstrated mechanisms for expression of a recessive phenotype at an autosomal locus are similar to those found in retinoblastoma and other tumors. Thus, APRT may be used to model loci important to carcinogenesis. (Full text available from University Microfilms International, Ann Arbor, MI, as Order Number AAD92-31641.)

L21 ANSWER 30 OF 46 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 1981:132626 BIOSIS
DN PREV198171002618; BA71:2618
TI **MUTATION** OF CHINESE HAMSTER CELLS BY NEAR UV ACTIVATION OF PRO MUTAGENS.
AU BARNHART B J [Reprint author]; COX S H
CS GENETIC TOXICOL MICROBIOL SECTION, MIDWEST RES INST, 425 VOLKER BOULEVARD, KANSAS CITY, MO 64110 USA, USA
SO Mutation Research, (1980) Vol. 72, No. 1, pp. 135-142.
CODEN: MUREAV. ISSN: 0027-5107.
DT Article
FS BA
LA ENGLISH
AB A tissue-**culture assay** for mutagenesis and cytotoxicity incorporating near near (NUV) light activation of polyaromatic hydrocarbons (PAH) was developed. **Cultures** of Chinese hamster ovary (CHO) cells growing in suspension **culture** were inoculated with benzo[a]pyrene (B[a]P), 7,12-dimethylbenzanthracene (DMBA) or shale-oil retort-water and exposed to light from a high-pressure mercury lamp fitted with a Corning NUV bandpass filter. This light source permitted activation of PAH and the shale-oil water and precluded detectable damage to DNA. Neither the PAH nor the NUV alone had any effect on cell survival or **mutation** frequencies, but the chemicals plus NUV were extremely effective in producing mutations to 6-thioguanine resistance due to mutations at the hgprt [hypoxanthine guanine phosphoribosyltransferase] **gene**.

L20 ANSWER 2 OF 9 MEDLINE on STN
AN 90081356 MEDLINE
DN PubMed ID: 2687628
TI Summary of complementation groups of UV-sensitive CHO cell **mutants** isolated by large-scale screening.
AU Busch D; Greiner C; Lewis K; Ford R; Adair G; Thompson L
CS Department of Environmental and Drug-Induced Pathology, Armed Forces Institute of Pathology, Washington, DC 20306-6000.
NC CA04484 (NCI)
GM22021 (NIGMS)
RR00961 (NCRR)
SO Mutagenesis, (1989 Sep) 4 (5) 349-54.
Journal code: 8707812. ISSN: 0267-8357.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199001
ED Entered STN: 19900328
Last Updated on STN: 19970203
Entered Medline: 19900125
AB A summary is given for the lineage and complementation group assignments of 153 UV-sensitive **mutants** of the CHO AA8 cell line. The distribution of **mutants** among six complementation groups was highly non-random, with the great majority of the isolates belonging to groups 1 and 2. This asymmetry is consistent with the known hemizygosity of these two linked loci in CHO cells. The relative numbers of **mutants** induced in group 2 was found to depend greatly on the type of mutagen used. Mutagenesis with UV radiation, ethyl methanesulfonate (EMS), N-methyl-N'-nitro-N-nitrosoguanidine and 7-bromomethylbenz[a] **anthracene** produced high frequencies of group 2 **mutants**. In contrast, ICR170 and ICR191, which are thought to produce mostly frameshift mutations, yielded very few **mutants** in group 2. These results are of particular importance in light of the recent finding that the human ERCC2 **gene**, which corrects group 2 **mutants**, has very strong homology with the **yeast** **gene** RAD3. RAD3 is an essential **gene** for viability in **yeast**, and the low recovery of group 2 **mutants** using the frameshift agents strongly suggests that frameshift mutations tend to be lethal in the hamster ERCC2 locus. Several mutagen-sensitive double **mutants** were isolated in two-step selections from EMS-, mitomycin C- or UV-sensitive parental cells, including the line UVU1, the first mammalian line with two mutations that affect UV sensitivity. The first **mutation** inactivated excision repair, and the second **mutation** appears to have affected some other recovery process. UVU1 should be useful for studying recovery processes that are separate from nucleotide excision repair.

L21 ANSWER 35 OF 46 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
AN 1998255917 EMBASE
TI Use of primary rat and human hepatocyte sandwich **cultures** for activation of indirect carcinogens: Monitoring of DNA strand breaks and **gene** mutations in co-**cultured** cells.
AU Fahrig R.; Rupp M.; Steinkamp-Zucht A.; Bader A.
CS R. Fahrig, Fraunhofer-Institute for Toxicology, Aerosol Research, Department of Genetics, Nikolai-Fuchs-Strasse 1, D-30625 Hannover, Germany
SO Toxicology in Vitro, (1998) 12/4 (431-444).
Refs: 30
ISSN: 0887-2333 CODEN: TIVIEQ
PUI S 0887-2333(98)00005-8
CY United Kingdom
DT Journal; Article
FS 052 Toxicology
LA English
SL English
AB Loss of cytochrome P-450 content is a common feature in conventional **culture** systems of primary hepatocytes. In contrast to the standard in **vitro** situation, in vivo each hepatocyte is exposed to an extracellular matrix (space of Disse) at two opposing basolateral surfaces. This in vivo symmetry has been reconstructed in **vitro** by **culturing** rat or human hepatocytes within two layers of collagen, thus forming a sandwich configuration. Activation of dimethylbenzanthracene (DMBA) or benzo[a]pyrene (BaP) was studied in rat and human hepatocytes. Genotoxic effects were studied in a three-dimensional co- **culture** model between sandwich hepatocytes and mammalian cells using the comet **assay** for detection of DNA strand breaks, and the HPRT test for detection of **gene** mutations. Sandwich hepatocytes generated active metabolites. The maintenance of metabolic properties in hepatocytes was dependent on extracellular matrix geometry. The number of DMBA- or BaP- induced genotoxic effects tended to be higher than in standard S-9 mix **assays**. While the ability to activate indirect carcinogens disappears within hours in primary hepatocytes, hepatocyte sandwich **cultures** enhance their ability to activate indirect carcinogens within 1 wk and retain this activity for up to 2 wk. This is the main advantage of the sandwich method over the more simple and conventional **assays**. While freshly isolated hepatocytes, regardless of whether in sandwich **culture** or in conventional **assays**, are injured by the isolation procedure and possess a corresponding reduced activation ability, hepatocytes in sandwich **cultures** recover over the course of a few days, and acquire a much higher ability to activate indirect carcinogens. Consequently, the indirect carcinogens BaP and DMBA, which were ineffective (BaP) or exhibited only weak effects (DMBA) at a concentration of 160 nmol/ml in 1-2-day-old hepatocytes, were clearly effective (BaP) or showed about a threefold increase in genotoxicity (DMBA) in 8-day-old hepatocytes in sandwich co-**culture**. In contrast to the experiments with S-9 mix, which is toxic to mammalian cells and does not allow treatment times of more than 2-3 hr, cells in co-**culture** with human or rat hepatocytes can be treated for at least 24 hr. The use of sandwich **cultures** has not yet been described for genotoxicity studies. The results of the present study may perhaps facilitate the acceptance of this method as a co-**culture** model for the field of genetic toxicology. Use of hepatocytes alone for genotoxicity studies cannot be recommended for difficulties in isolating intact cells from the sandwich **cultures** . The use of human hepatocytes in sandwich co-**culture** should enable a more relevant evaluation of potential human genotoxicity with specific chemicals and should put the extrapolation of genetic toxicology data from animal species to humans on a more scientific basis. Beyond that, experiments with animals in vivo could be avoided.

L21 ANSWER 39 OF 46 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
AN 92237889 EMBASE
DN 1992237889
TI C-Ha-ras **gene mutation** and activation precede pathological changes in DMBA-induced in vivo carcinogenesis.
AU Kwong Y.Y.; Husain Z.; Biswas D.K.
CS Laboratory of Molecular Biology, Harvard School of Dental Medicine, 188 Longwood Avenue,Boston, MA 021115, United States
SO Oncogene, (1992) 7/8 (1481-1489).
ISSN: 0950-9232 CODEN: ONCNES
CY United Kingdom
DT Journal; Article
FS 005 General Pathology and Pathological Anatomy
016 Cancer
022 Human Genetics
LA English
SL English
AB We have previously reported a stage-specific and sequential overexpression of the c-Ha-ras and c-erbB genes in 7, 12-dimethylbenzanthracene (DMBA)-induced in vivo carcinogenesis in hamster buccal pouch epithelium (HBPE). In this investigation, the immunoreactive protein product of the c-Ha-ras **gene** (p21 protein) was **identified** in HBPE cells, specifically in treated tissues and **cultured** cells established after 3 weeks of DMBA treatment. Microscopic examination did not show any histopathological changes in these tissues. The p21 protein was detected in a few selective cells, which were dispersed away from the more densely populated basal layer. The overexpression of the c-Ha-ras **gene** was accompanied by a point **mutation** of A→T in codon 61 (CAA), inducing an amino acid substitution from the wild-type glutamine to leucine in the peptide. The concurrent molecular modifications preceded any detectable histopathological changes. The cellular morphology and orientation in treated HBPE at this early stage was indistinguishable from the control tissue. Yet the genetic alterations, such as the point **mutation** and overexpression of the **gene**, were evident at the predysplastic stage. Amplification and overexpression of the second protooncogene, c-erbB, and its product, epidermal growth factor receptor (EGFR), were detected in HBPE cells at the later stages of extensive cell proliferation and invasion. By using double antibodies and two immunoreporter systems, we demonstrated overexpression of both c-Ha-ras and c-erbB genes in the same HBPE cells during this chemically induced in vivo carcinogenesis.

L21 ANSWER 41 OF 46 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
AN 87231439 EMBASE
DN 1987231439
TI Analysis of the ras(H) oncogene and its p21 product in chemically induced skin tumors and tumor-derived cell lines.
AU Harper J.R.; Reynolds S.H.; Greenhalgh D.A.; Strickland J.E.; Lacal J.C.; Yuspa S.H.
CS Laboratory of Cellular Carcinogenesis and Tumor Promotion, National Cancer Institute, Bethesda, MD, United States
SO Carcinogenesis, (1987) 8/12 (1821-1825).
ISSN: 0143-3334 CODEN: CRNGDP
CY United Kingdom
DT Journal
FS 013 Dermatology and Venereology
016 Cancer
022 Human Genetics
LA English
AB Mouse skin papillomas and squamous cell carcinomas induced by initiation with 7,12-dimethylbenz[a]**anthracene** and promotion with phorbol esters, such as 12-O-tetradecanoyl phorbol-13-acetate, frequently contain an activated Harvey ras **gene**. Six murine epidermal cell lines established from pooled skin papillomas previously tested negative in the NIH-3T3 **assay**, but have an altered differentiation program by a variety of criteria. The Harvey ras **gene** and its p21 protein product from these cell lines have been analyzed for alterations responsible for their altered growth and differentiation properties that were undetectable by 3T3 transfection **assays**. In comparison with primary papillomas and carcinomas, shown to have a point **mutation** in codon 61 of the Harvey ras **gene**, resulting in a p21 product with the diagnostic alteration in SDS-PAGE, the papilloma cell lines exhibited neither the codon 61 **mutation**, nor p21 product with altered migration in SDS-PAGE. These findings suggest that these papilloma cell lines contain a genetic lesion(s), other than Harvey ras activation, that may be responsible for their alterd epithelial differentiation patterns and thus may serve as a useful model for **identifying** lesions involved in malignant conversion.

L21 ANSWER 38 OF 46 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
AN 95323882 EMBASE
DN 1995323882
TI Stereoselectivity of activation of 7,12-dimethylbenz[a]**anthracene** -3,4- dihydrodiol to the anti-diol epoxide metabolite in a human mammary carcinoma MCF-7 cell-mediated V79 cell **mutation assay**.
AU Lau H.H.S.; Coffing S.L.; Lee H.; Harvey R.G.; Baird W.M.
CS Department of Medicinal Chemistry, Hansen Building, Purdue University,West Lafayette, IN 47907, United States
SO Chemical Research in Toxicology, (1995) 8/7 (970-978).
ISSN: 0893-228X CODEN: CRTOEC
CY United States
DT Journal; Article
FS 052 Toxicology
LA English
SL English
AB 7,12-Dimethylbenz[a]**anthracene** (DMBA), one of the most carcinogenic polycyclic aromatic hydrocarbons in rodent bioassays, is metabolically activated in many tissues to 'bay-region' DMBA-3,4-diol-1,2-epoxides (DMBADE). Unlike benzo[a]pyrene, for which the high biological activity of the (7R,8S)-diol-(9S,10R)-epoxide has been established, the low chemical stability of anti-DMBADE has made it impossible to evaluate the role of specific stereoisomers in the biological activity of DMBA. In order to characterize the role of formation of DMBADE diastereomers in the induction of mutations, postlabeling **assays** using [35S]phosphorothioate with adduct separation by HPLC and immobilized boronate chromatography analyses were developed to allow separation and quantitation of DNA adducts formed from each stereoisomer of DMBADE. In DMBA-treated hamster embryo cell **cultures**, large quantities of three major adducts (anti-DMBADE-deoxyguanosine, anti- DMBADE-deoxyadenosine, and syn-DMBADE-deoxyadenosine) along with five minor adducts were completely resolved and quantitated. The DNA isolated from a human mammary carcinoma MCF-7 cell-mediated V79 cell **mutation assay** treated with increasing doses of racemic DMBA-3,4-dihydrodiol contained large amounts of two anti-DMBADE-DNA adducts. The anti-DMBADE adducts accounted for more than 90% of the total adducts at all doses. The number of 6-thioguanine- resistant **mutants** was proportional to the amount of anti-DMBADE-DNA adducts. The results demonstrate that the MCF-7 human mammary tissue carcinoma cell line stereoselectively activates DMBA-3,4-dihydrodiol to mutagenic anti- DMBADE and indicate that human cells can effectively activate bay-region diols of hydrocarbons containing a methyl-hindered bay region, a structural feature frequently found in highly carcinogenic polycyclic aromatic hydrocarbons.

L21 ANSWER 24 OF 46 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1993:74860 CAPLUS
DN 118:74860
TI Detection of mammalian carcinogens with an immunological DNA synthesis-inhibition test
AU Heil, Juergen; Reifferscheid, Georg
CS Dep. Environ. Mol. Genotoxicity, Univ. Mainz, Mainz, 6500, Germany
SO Carcinogenesis (1992), 13(12), 2389-94
CODEN: CRNGDP; ISSN: 0143-3334
DT Journal
LA English
AB The Salmonella **gene mutation assay** (Ames test) is the most widely used test for the screening of mutagens. However, many in **vitro** tests hold unsatisfactory validity data, presumably because of the inability of present short-term tests to detect nongenotoxic carcinogens, which are increasingly being brought into focus in the discussions of genesis of cancer. One principle often neglected in this context is the property of genotoxic agents to inhibit replicative DNA synthesis in (proliferating) eukaryotic cells. The authors believe that this early response to DNA damage is important in the multistage process of carcinogenesis. Accordingly, the authors proposed that a DNA synthesis-inhibition test should be included in the test batteries for carcinogen screening. The development of an appropriate DNA synthesis-inhibition test based on immunol. techniques is reported.

L20 ANSWER 1 OF 9 MEDLINE on STN
AN 2001047126 MEDLINE
DN PubMed ID: 10943946
TI Establishment and characterization of a cell line (HCDB-1) derived from a hamster buccal pouch carcinoma induced by DMBA and Taiwanese betel quid extract.
AU Lin S C; Chang K W; Chang C S; Yu S Y; Chao S Y; Wong Y K
CS Institute of Oral Biology and Department of Dentistry, National Yang-Ming University, Taipei, Taiwan, ROC.
SO Proceedings of the National Science Council, Republic of China. Part B, Life sciences, (2000 Jul) 24 (3) 129-35.
Journal code: 8502426. ISSN: 0255-6596.
CY CHINA (REPUBLIC: 1949-)
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200012
ED Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20001204
AB This study identified that the carcinogenesis of hamster buccal pouch (HBP) induced by 7,12-dimethylbenz[a]**anthracene** (DMBA) was greatly enhanced (18 folds) by a combination treatment with Taiwanese betel quid (BQ) extract. A new cell line, HCDB-1, has been established from induced carcinomas. The **cultured** monolayer cells were epithelioid in shape with irregular nuclei. They demonstrated abundant cytokeratin and tonofilaments; however, ultrastructural well-organized desmosomes were lacking. The HCDB-1 cell exhibited population doubling in 19 h and was highly tumorigenic in nude mice. A C-->T transition at codon 141 (Ala to Val) of the p53 **gene** was detected in this cell. This **mutation** is equivalent to a specific temperature-sensitive mouse p53Ala135Val **mutant** that causes transformation by shifting to 37.5 degrees C. HCDB-1 is the first cell line established from the HBP model of oral carcinogenesis induced by DMBA/Taiwanese BQ extract. It might be valuable for exploring the molecular pathogenesis of oral cancer.

L6 ANSWER 11 OF 23 MEDLINE on STN
AN 1999165869 MEDLINE
DN PubMed ID: 10064862
TI Repair of DNA lesions: mechanisms and relative repair efficiencies.
AU Braithwaite E; Wu X; Wang Z
CS Graduate Center for Toxicology, University of Kentucky, 306 Health Sciences Res. Building, Lexington, KY 40536-0305, USA.
NC CA67978 (NCI)
ES5796 (NIEHS)
SO Mutation research, (1999 Mar 8) 424 (1-2) 207-19.
Journal code: 0400763. ISSN: 0027-5107.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199905
ED Entered STN: 19990517
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AB DNA is frequently damaged by endogenous agents inside the cells. Some exogenous agents such as polycyclic aromatic hydrocarbons (PAHs) are ubiquitous in the environment and may thus contribute to the 'background' DNA damage in humans. DNA lesions are normally removed by various repair mechanisms. The major repair mechanisms for various DNA lesions are summarized. In contrast to the extensively studied repair mechanisms, much less is known about the relative repair efficiencies of various DNA lesions. Since DNA repair is a crucial defense against carcinogenesis, it may constitute an important factor affecting the carcinogenicity of DNA damaging agents. We have adopted a human cell-free system for measuring relative DNA repair efficiencies based on the concept of repair competition between acetylaminofluorene adducts and other DNA lesions of interest. Using this in vitro system, we determined the relative repair efficiencies of PAH adducts induced by: anti-(+/-)-benzo[a]pyrene-trans-7,8-dihydrodiol-9,10-epoxide (BPDE), anti-(+/-)-benz[a]**anthracene**-trans-3,4-dihydrodiol-1,2-epoxide (BADE-I), anti-(+/-)-benz[a]**anthracene**-trans-8,9-dihydrodiol-10, 11-epoxide (BADE-II), anti-(+/-)-benzo[b]fluoranthene-trans-9, 10-dihydrodiol-11,12-epoxide (BFDE), anti-(+/-)-chrysene-trans-1, 2-dihydrodiol-3,4-epoxide (CDE), and anti-(+/-)-dibenzo[a, l]pyrene-trans-11,12-dihydrodiol-13,14-epoxide (DBPDE). While damage by BPDE, DBPDE, CDE, and BFDE were repaired by nucleotide excision repair as efficiently as AAF adducts, the repair of BADE-I and BADE-II adducts were significantly slower in human cell extracts. Damage by DBPDE at 3 microM in vitro yielded approximately 5-fold higher DNA adducts than BPDE as determined by quantitative PCR. This potent DNA reactivity may account in part for the potent carcinogenicity of dibenzo[a,l]pyrene. The correlation of these results to the carcinogenic properties of the PAH compounds is discussed. Furthermore, we show that NER plays a role in AP site repair in vivo in the eukaryotic model organism **yeast**.
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